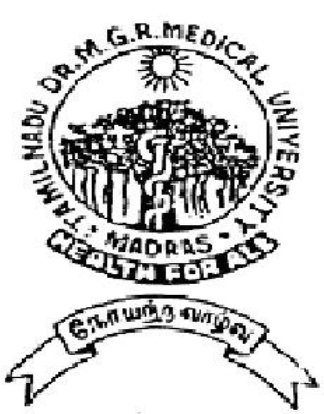
# A DISSERTATION ON HEARING ASSESSMENT OF AT-RISK NEONATES

*Dissertation Submitted for*

**MD Degree (Branch VII) PEDIATRICS**

**April 2011**

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# CERTIFICATE

This is to certify that the dissertation entitled **"HEARING ASSESSMENT OF AT-RISK NEONATES"** submitted by **Dr.R.VENKATARAMANAN** to the faculty of Paediatrics, The Tamil Nadu Dr.M.G.R Medical University,Chennai in partial fulfillment of the requirement for the award of M.D. Degree Branch VII (Paediatrics) is a bonafide research work carried out by him under our direct supervision and guidance.


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# INTRODUCTION

Hearing screening at birth has been omitted by most of the centers in India at the moment. The incidence of congenital hearing loss in the newborn population is greater than the combined incidence of all the metabolic conditions that we currently screen for with blood tests.[26] Significant hearing loss is one of the most major abnormalities present at birth About one in every thousand children is born profoundly deaf,four times as many are born with moderate or severe bilateral hearing loss.[18] Infants in Neonatal intensive care units are 10 – 20 times more likely to have significant hearing loss than healthy population.

In 1993 a consensus statement from the National Institute of Health (NIH) recommended universal newborn hearing screening by the age of 3 months and also stated that otoacoustic emission might be the technology used for screening[27].

These recommendations were based on the following: (1) the incidence of hearing loss is 1 to 6 per 1000; (2) only one half of the infants with hearing loss are discovered with high-risk screening; (3) the current

average age at diagnosis of hearing loss is 2.5 years; and (4) early identification and treatment by the age of 6 months will improve outcomes.[8]

As the first year of life is substantially critical in the development of brain, absence of auditory experience during this period significantly retards the child's overall development. Thus there is a need for early identification of hearing loss through Newborn Hearing Screening Programmes which is already existing in developed countries.[10]

# REVIEW OF LITERATURE

## EPIDEMIOLOGY

According to the current estimates permanent hearing loss of greater than 25 decibel hearing level in poorer ear is present in atleast 4(1.1 to 6) per 1000.[16] As many as half of these infants have no risk factors for hearing loss; thus hearing loss may not be suspected until the child misses language milestones. The prevalence of hearing loss in high risk infants is estimated to be between 2.5 and 10%.[20,23]. Newborn hearing program aims to detect hearing impairment including unilateral or bilateral sensory or conductive hearing loss averaging 30 – 40 decibel or more in frequency region 500 through 4000 HZ. Hearing impairment in this range will have most impact on speech acquisition[27].

In India, incidence of hearing impairment in neonates ( at risk and not at risk) ranges from 6 – 60 per 1000 neonates with an average of 4 per 1000 neonates.[4] Another survey in India has shown 4 out of every 1000 infants born were found to have severe hearing loss.[21]

## NEED FOR HEARING ASSESSMENT IN NEWBORN

The first three years of life are most important for language and speech development. Consequently, for many infants and young children with unidentified hearing impairment much of the crucial period for language and speech development may be lost. Moderate to profound hearing loss in early infancy has been shown to be associated with impaired language development, as auditory stimuli during this period are critical to development of speech and language skills[27].

This in turn leads to lower reading abilities, poor academic achievement and fewer career opportunities (Task Force on newborn and infant hearing).

The personal and social impacts of hearing loss are enormous. People with hearing impairment "often have less desirable jobs and incomes than people without hearing impairment." Lifetime costs of each case of congenital deafness have been estimated at over $1 million, and "programs and services for the communicatively handicapped are estimated to cost $23.4 billion per year in the United States." Costs of lost earnings to people resulting from the disability caused by hearing loss was estimated to be over $1.25 billion annually in 1990. Other burdens arise because of "emotional

stress, breakdowns in family communication, and isolation of hearing impaired persons from peers and educational systems."

A child's hearing impairment should therefore be identified as early in life as possible so that he or she can receive timely and appropriate intervention. The interventions will then take full advantage of the plasticity of child's developing nervous system optimizing his or her social, emotional, psychological and academic development.[11,22,25]

In 1993,the NIH recommended that all children should be screened for hearing loss by 3 months. In 1994,Joint committee on infant hearing(JCIH) endorsed this recommendation and suggested that screening should take place before a newborn was discharged from the hospital where they were born to ensure most of the children could be screened.

Universal newborn screening for hearing loss began in January 1, 2003. The goals are detection of hearing loss before 3 months of age , and the initiation of appropriate intervention no later than 6 months of age. American Academy of Paediatrics(AAP) and Joint Committee on Infant Hearing(JCIH) also recommend the same. The Early Hearing Detection and Intervention (EHDI) Program of the Center for Disease Control and Prevention(CDC) also recommends the "1-3-6 plan," ie all infants to be screened by age **1** month, all children who do not pass the screening to

receive diagnostic audiological testing by age **3** months, and all children with confirmed hearing loss to be enrolled in an appropriate intervention program by the age of **6** months.[25]

Prior to universal newborn hearing screening implementation,average age of identification of hearing loss was 20.2 months.Two years after universal newborn hearing screening implementation,average age of diagnosis has greatly improved to 3.8 months of age.[17,22]

**DEVELOPMENT OF EAR**[3]

**AURICLE –** About the 6th week of embryonic life, a series of 6 tubercles appear around the first branchial cleft.They coalesce to form the auricle. Tragus develops from the tubercle of first arch while rest of pinna develops from the remaining five tubercles of the second arch.

**EXTERNAL AUDITORY MEATUS –** It develops from the first branchial cleft.By 16th week of embryonic life, cells proliferate from the bottom of ectodermal cleft and form a meatal plug. Recanalisation of this plug forms the epithelial lining of the bony meatus.

**TYMPANIC MEMBRANE** – It develops from all the 3 germinal layers.

Outer epithelial layer is formed by the ectoderm, inner mucosal layer by the endoderm and the middle fibrous layer by the mesoderm.

**MIDDLE EAR CLEFT** – The Eustachian tube, tympanic cavity, attic, antrum and mastoid air cells develop from the first and second pharyngeal pouches.Malleus and Incus are derived from mesoderm of the first arch while the Stapes from the second arch.

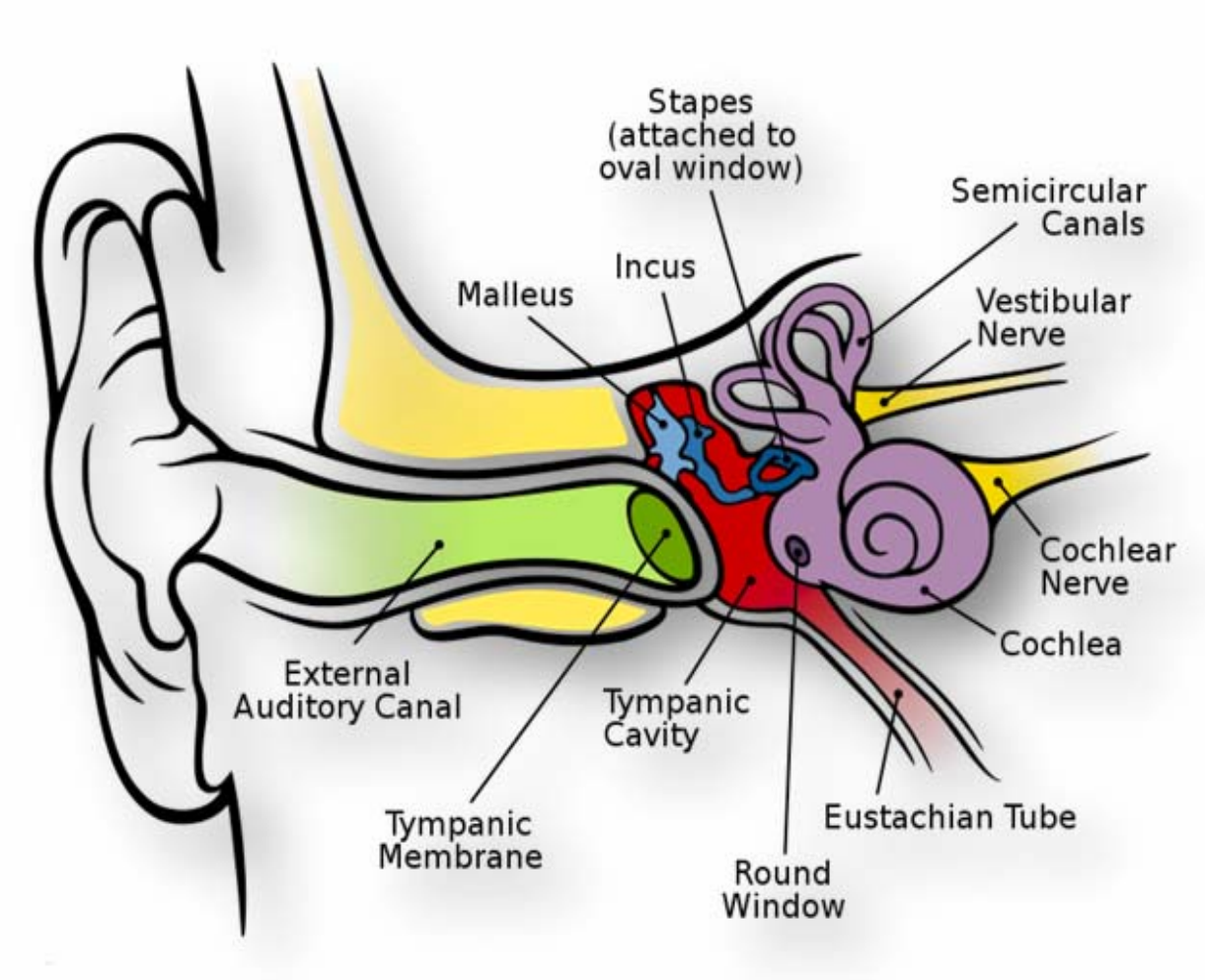
**INNER EAR** –Ectoderm in the region of hind brain thickens to form an auditory placode which is invaginated to form the auditory vesicle or otocyst.This differentiates into the endolymphatic duct and sac; the utricle,semicircular ducts; and saccule and the cochlea

**TABLE – 1**

**TIMING OF DEVELOPMENT OF EAR IN THE WEEK OF GESTATION**

| Development | Pinna | Meatus | Middle Ear | Vestibular Labyrinth | Cochlea |
|---|---|---|---|---|---|
| BEGINS | $6^{th}$ | $8^{th}$ | $3^{rd}$ | $3^{rd}$ | $3^{rd}$ |
| COMPLETES | $20^{th}$ | $28^{th}$ | $30^{th}$ | $20^{th}$ | $20^{th}$ |

## ANATOMY OF EAR



**MECHANISM OF HEARING**

A sound signal in the environment is collected by the pinna, passes through the external auditory canal and strikes the tympanic membrane.

Vibrations of the tympanic membrane are transmitted to the stapes footplate through a chain of ossicles coupled to the tympanic membrane.

Movements of the stapes footplate cause pressure changes in the labyrinthine fluids which move the basilar membrane.This stimulates the hair cells of organ of corti.It is the hair cells which convert mechanical energy into electrical impulse

Hair cells are innervated by dendrites of bipolar cells of spiral ganglion.Axons of these bipolar cells form the cochlear division of $8^{th}$ cranial nerve which enters the brain at ponto medullary junction.

On entering the brainstem,fibres bifurcate.The upper division ends in **DORSAL COCHLEAR NUCLEUS (DCN) BILATERALLY**.The lower division ends in **VENTRAL COCHLEAR NUCLEUS(VCN).**

II order neurons from DCN ascend in **LATERAL LEMNISCUS** while II order neurons from VCN relay in **SUPERIOR OLIVARY NUCLEUS**. From the superior olivary nucleus, III order neurons ascend in the lateral lemniscus

↓

Lateral lemniscal fibres terminate in **INFERIOR COLLICULUS**. Intercollicular commissural fibers transmit impulses between the colliculi.

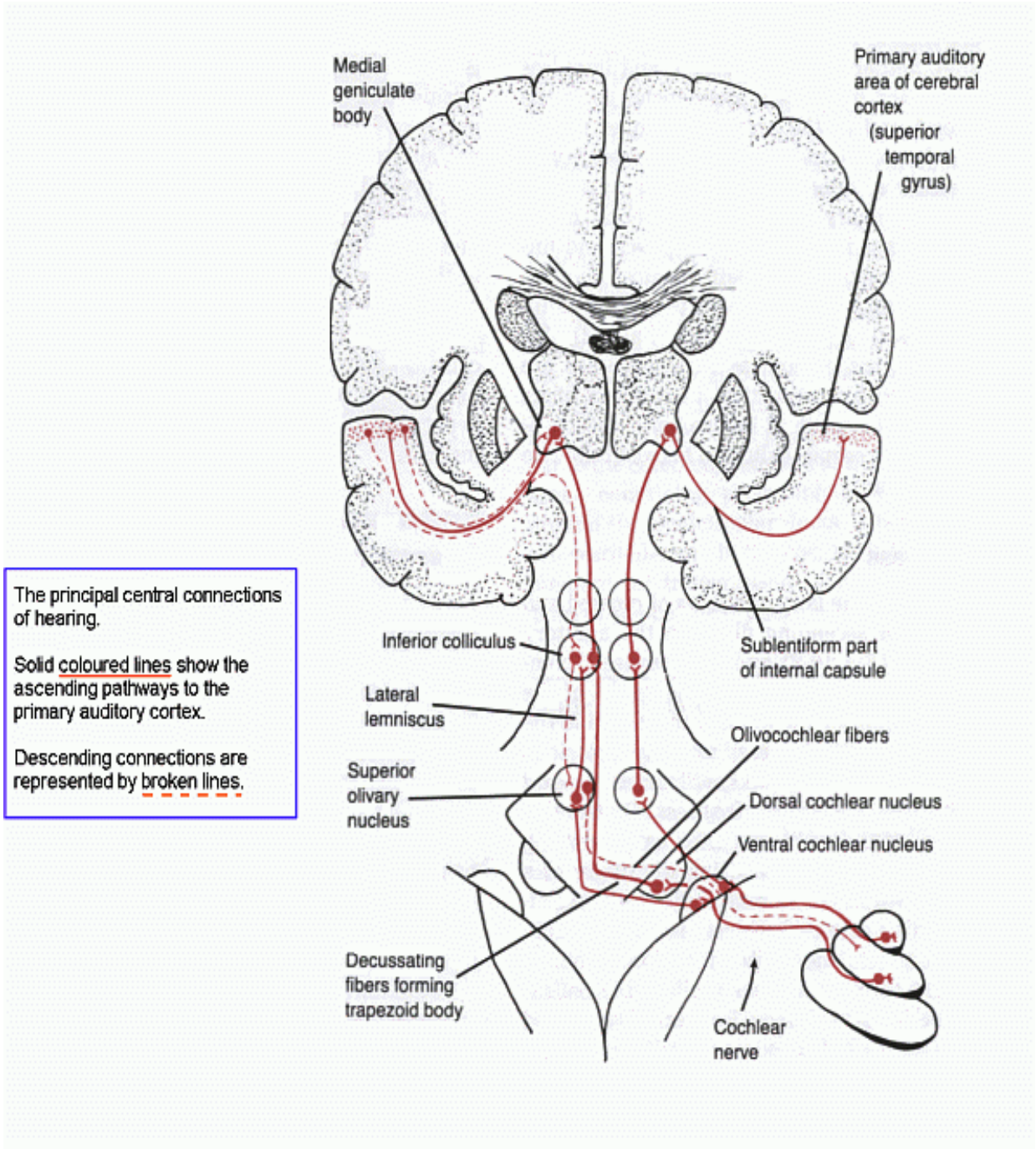
↓

From the inferior colliculus,impulses are projected into **IPSILATERAL MEDIAL GENICULATE BODY**

↓

From the medial geniculate body,impulses are projected to **AREA 41** or **HESCHL"S GYRUS**(Superior Temporal Gyrus) known as **Primary Auditory Cortex**.Some impulses are also projected to AREA 42, the Auditory Association Area.

# AUDITORY PATHWAY



## HEARING LOSS[2]

Hearing loss falls into **four** major categories :

- **Sensory neural loss** is the result of abnormal development or damage to the Cochlear hair cells or auditory nerve.
- **Conductive loss** is the result of interference in the transmission of sound from the external auditory canal to the inner ear.The most common cause for conductive hearing loss is fluid in the middle ear or middle ear effusion. Less common are anatomic causes such as microtia, canal stenosis,or stapes fixation that often occur in infants with craniofacial malformation.
- **Auditory dyssynchrony or auditory neuropathy** – Here the cochlea receives sounds normally ; however the transfer of signal from the cochlea to the auditory nerve is abnormal.The etiology of this condition is not understood ; however,babies who have severe hyperbilirubinemia,prematurity,hypoxia and immune disorders are at increased risk.
- **Central hearing loss** – In this type of hearing loss there is an intact auditory canal and inner ear and normal neurosensory pathways but

abnormal auditory processing at higher levels of the central nervous system.

## HEARING LOSS - CAUSES

The causes fall into three basic categories.

- **Genetically** inherited hearing loss accounts for approximately 50% of all cases. 70 % are autosomal recessive,15% autosomal dominant and 15% with other types of transmission.The most common genetic cause of hearing loss is a mutation in the **connexin 26 gene**,located on chromosome **13q11-12.**Deletion of **mitochondrial gene 12SrRNA,A1555G** is associated with a predisposition for hearing loss after exposure to aminoglycoside antibiotics. The majority of these are non-syndromic. The other cases are syndromic. In this type there are other clinical manifestations along with the hearing loss. Usher syndrome is an example of a syndrome that includes hearing loss.

- In approximately 25% of childhood hearing loss,a **nongenetic** cause is identified. Hearing loss is thought to be secondary to injury to the developing auditory system in the intrapartum or perinatal period.The

injury may be due to infection, hypoxia, ischaemia, metabolic disease, ototoxic medication or hyperbilirubinemia. Congenital Cytomegalovirus infection is the most common cause of nonhereditary sensoryneural hearing loss.Approximately 1% of all infants are born with CMV infection.Of these 10% have clinical signs of infection at birth(small for gestational age, hepatosplenomegaly, jaundice, thrombocytopenia, neutropenia, intracranial calcification, skin rash) and 50 -60% of these infants develop hearing loss.Hearing loss also develops in 10 – 15% of those who are asymptomatic at birth. Sensoryneural deafness is the single most common finding among infants with Congenital Rubella Syndrome(when maternal infection occurs before 11 weeks of gestation).

- In the remaining 25% there is no identifiable cause.

**TABLE-2**

**SEVERITY OF HEARING LOSS**

| | |
|---|---|
| MILD | 15 -30 dB HEARING LOSS |
| MODERATE | 30 -50 dB HEARING LOSS |
| SEVERE | 50 -70 dB HEARING LOSS |
| PROFOUND | 70+ dB HEARING LOSS |

## SCREENING METHODS

Previously, the standard hearing test was behavioral assessment (Murphy’s Sound localization method).Under this technique, the infant would be subjected to a sound while observer watches a reaction from the baby in response to it (i.e., testing an infant's "startle response"). The method is often limited by the observer's ability to subjectively assess the infant's reaction to the sound at the time of the test.

Over the last two decades, more sophisticated measures have been devised and used to measure physiologic changes in the baby arising in response to sound. Two primary methods are now used to check newborn hearing. Otoacoustic emission (OAE) testing uses a probe placed within the infant's ear to measure inner-ear responses to sound. Automated Auditory brainstem response (AABR) uses electrodes placed on the infant's head to measure brain-wave responses to clicks administered to the ear. These methods are more accurate for infants under 6 months of age than behavioral assessment.These tests have been recommended by AAP and JCIH.Both hearing screening methods are objective and physiologic measures that do not need the active patient response required in traditional audiologic evaluation tests[27].

Hearing screening tests provide a quick and cost effective way to separate people into two groups: a pass group and a fail group. Those who pass hearing screening are presumed to have no hearing loss. Those who fail are in need of an in-depth evaluation by an audiologist.

All of the tests described above are safe and non-invasive.Some skin abrasions from the electrodes are the only complications associated with AABR; no complications are reported from OAE testing. Otoacoustic emission testing can be done with the infant awake, feeding, or sucking on a pacifier. AABR requires the infant to be asleep.

There are 3 steps in the Hearing Screening program.

- **Screening**
- **Confirmation** (Audiologic evaluation of those with abnormal result) and
- **Early intervention** for those with confirmed hearing impairment.

**OTOACOUSTIC EMISSIONS(OAE)**

Oto acoustic emissions were first described by KEMP in 1978.In healthy cochlea,vibration of hair cells in response to noise generates acoustic energy known as OAE.An OAE is a weak echo type inaudible sound emitted

by the ear soon after an audible sound is perceived.The OAE measures stimulated acoustic energy generated in cochlea (inner ear) that travels through the middle ear into the ear canal where it is sensed with a miniature microphone. OAE is very sensitive, noninvasive, cost and time effective making it an ideal screening method.[26]

OAE testing therefore measures the integrity of inner ear. Persons with normal hearing produce emissions. Those with hearing loss greater than 25 – 30 decibels do not. OAEs can detect blockage in outer canal,middle ear fluid or damage to outer hair cells in the cochlea.

To perform the OAE,a tiny flexible plug is inserted into baby's ear.Specific sounds are generated through the plug.A miniature microphone in the plug records the otoacoustic responses of the inner ear in reaction to transmitted sounds.The test is usually done when the baby sleep.

Automated OAE screeners display the results of the test as either 'PASS' or 'REFER'requiring no interpretation by screening personnel. Refer means either the ear is abnormal or there is false positive result due to debris in the external canal. This test takes between 1-5 minutes to perform.

# OAE INSTRUMENT

## AUTOMATED AUDITORY BRAINSTEM RESPONSE(AABR)

The AABR provides complete screening of auditory pathway upto the brainstem(including middle ear, inner ear and 8$^{th}$ N). When AABR is performed electrodes are placed on the forehead, nape of neck and shoulder(ground). With AABR screening ,a click stimulus at one loudness level is provided to each of the child's ears.The child's response is compared to a template of children with normal hearing. If the responses match, the child passes the screening; If they do not match then child has hearing impairment. Screening by AABR can be completed after birth and a stringent statistical pass criterion is employed that eliminates bias from interpretation.AABR is a screening tool for infant who have reached atleast 34 weeks conceptional age until the child turns 6 months of age.[12]

Responses from a large numbers of stimulus presentations are averaged and the automated screener uses a response algorithm to produce a PASS or REFER result.The pass level is set at about 35 decibels Babies are sedated to minimize electrical interference caused by muscle activity during testing. In a normal person 7 waves are produced in the first 10 milliseconds. Waves I,III and V can be obtained consistently in all age groups.Waves II and IV appear less consistently.The latency of each

wave(time of onset of wave peak after stimulus onset) increases and the amplitude decreases with reduction in stimulus intensity or loudness.[1,3]

The exact anatomic site of origin of waves is still disputed but they are thought to arise from following parts.

**TABLE – 3**

**SITE OF ORIGIN OF WAVES**

| | |
|---|---|
| WAVE I | VIII NERVE |
| WAVE II | COCHLEAR NUCLEI(PONS) |
| WAVE III | SUPERIOR OLIVARY COMPLEX(PONS) |
| WAVE IV | LATERAL LEMNISCUS(PONS) |
| WAVE V | INFERIOR COLLICULUS(MIDBRAIN) |
| WAVE VI | MEDIAL GENICULATE BODY(THALAMUS) |
| WAVE VII | AUDITORY RADIATIONS(THALAMO CORTICAL) |

**WHO SHOULD BE SCREENED?**

Ideally all infants have to be screened for congenital and neonatal onset hearing loss prior to the discharge from the hospitals where they were born. If this is not feasible due to financial constraints, then infants with the following **risk factors** should **DEFINITELY** be screened.[1,2,4,10,19]

1. An illness or condition requiring admission of 24 hours or more in NICU.
2. Birth weight less than 1,500 grams (3.3 lbs.).
3. Apgar scores of 0- 4 at 1 minute ; 0-6 at 5 minutes.
4. Hyperbilirubinemia at a serum level requiring exchange transfusion.
5. Ototoxic medications –Aminoglycosides and loop diuretics used for >5 days
6. Mechanical ventilation lasting 5 days or longer and PPHN.
7. Bacterial meningitis.
8. In utero infections by TORCH group of organisms.
9. Craniofacial anomalies, including those with morphological abnormalities of the pinna and ear canal.
10. Stigmata or other findings associated with a syndrome known to include a sensorineural and/or conductive hearing loss such as Waardenburg syndrome (pigmentary abnormalities),branchiootorenal

syndrome (ear tags or pits), Usher syndrome(retinitis pigmentosa), Pendred syndrome (thyroid enlargement), Jervell and Lange-Nielsen syndrome(cardiac conduction defects).

11. Family history of permanent childhood sensorineural hearing loss.

While high risk newborns do have much higher rates of hearing impairment, they account for only about 50 percent of all newborns with hearing loss at birth.

A consequence of screening only high risk neonates is that approximately only one in ten newborns is screened, and only about half of all hearing impaired infants are detected at birth.[18]

**WHAT HAPPENS IF AN INFANT DOES NOT PASS THE SCREENING?**[10,14,16,20]

Infants who do not pass the first screening by OAE are subjected to second screening by OAE after one month. Rescreening reduces the false positive rates[13,14].

If second screening is also abnormal,then the infant is subjected to AABR(Task Force on newborn and infant hearing) and then referred for **follow-up audiological (electrophysiologic measure of threshold using,**

**frequency specific stimuli) and medical evaluations that should occur no later than 3 months of age.** These evaluations confirm the presence of hearing loss; determine the type, nature, and (whenever possible) the cause of the hearing loss; and help to identify options for treatment. Intervention for hearing loss must be initiated before 6 months of age.

**SENSITIVITY AND SPECIFICITY**[26]

Sensitivity of OAE is 80 – 98% and that of AABR is 84-90%.

Both methods have specificity >90%.

**TEST LIMITATIONS**[26]

Both OAE and AABR require a quiet baby and a quiet testing environment.OAE relies on a functional outer, middle and inner ear and AABR in addition relies on functional lower auditory pathway.The screening tests are not designed to detect central hearing impairment (where there is hearing loss secondary to dysfunction of pathways from the brainstem to the auditory cortex).

As the stimuli for both tests are introduced via external ear canal,debris in the canal or middle ear fluid can affect accuracy of test.In particular, OAE testing may be affected by amniotic fluid in the ear canal when testing is done in the first 48 hours after birth.

## INTERVENTION FOR HEARING IMPAIRMENT

Numerous professionals are involved in offering services to a child with hearing impairment including.

- The Audiologist and audiological team
- Primary care Paediatrician
- Speech Therapist
- Otorhinolaryngologist
- Plastic Surgeon
- Alternative Language Teachers

## HEARING AIDS FOR CHILDREN

Fitting and programming hearing aids for infants and children should be done by a qualified paediatric audiologist.Both BTE (behind the ear) and ITE (in the ear) hearing aids are used to amplify sound for hearing impaired children. ITE hearing aids sit completely inside of the ear. While ITE hearing aids may be used by older children, they aren't recommended for infants and younger children.

During infancy and early childhood the size and shape of the ear changes as the child grows. BTE hearing aids are safer and can be utilized longer.Hearing aids may be fitted for infants as early as 2 months.


**IN THE EAR HEARING AID**

**COCHLEAR IMPLANTS**

In some situations hearing aids are not very effective. When there is profound sensorineural hearing loss,cochlear implants may be an option. A cochlear implant is an electronic device. There is an external component and surgically implanted internal component. The external component picks up the sound, converts it to electronic impulses and then relays the information

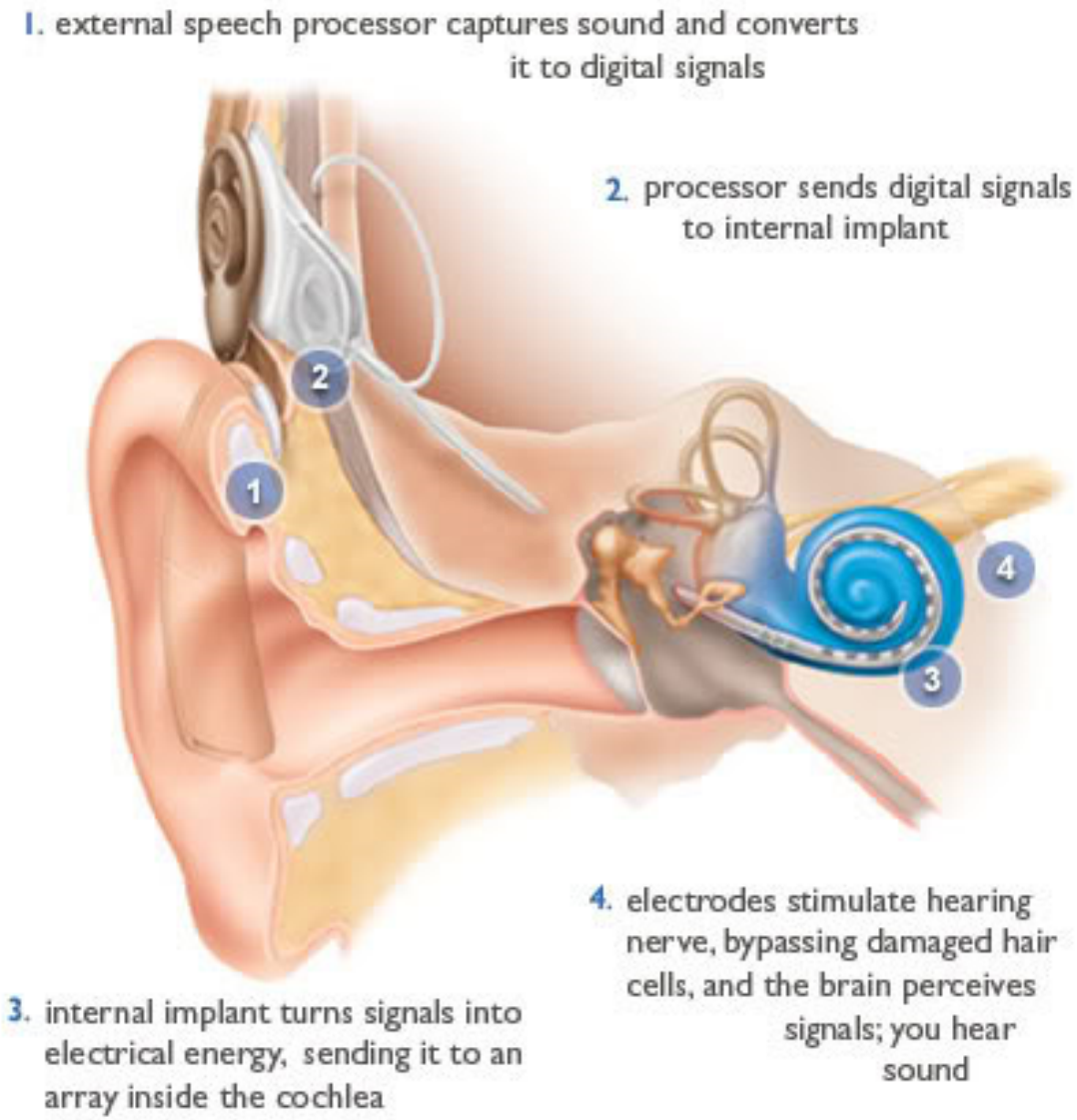
to the internal device. Impulses are collected by the receiver and then sent directly to the auditory nerve.

A serious complication of cochlear implants is an excessively high incidence of pneumococcal meningitis.All children receiving a cochlear implant must be vaccinated with the Pneumococcal vaccine.

**HOW A COCHLEAR IMPLANT WORKS?**

1. Sounds are picked up by the microphone.
2. The signal is then “coded”(turned into a specific pattern of electrical impulses).
3. These impulses are sent to the coil and are transmitted across the skin to the implant.
4. The implant sends a pattern of electrical impulses to the electrodes in the cochlea.
5. The Auditory nerve picks up the electrical impulses and sends them to the brain.The brain recognises these signals as sounds.

# COCHLEAR IMPLANT



**Nagapoornima et al**[4] – Conducted a prospective non-randomised study to determine the incidence of hearing impairment in at risk and not at risk neonates in tertiary care hospital in Bangalore.The incidence of hearing impairment was 7 out of 1490(NOT AT RISK) and 3 out of 279(AT RISK).

**Sharma et al**[12] – Conducted a study to evaluate BERA as an objective test of hearing assessment in icteric babies and concluded that BERA is a simple,reliable and effective technique for detection of hearing impairment in the neonates.

**Vaid et al** – Screened 2621 babies in a tertiary care hospital in Pune by OAE and BERA and found that 15 babies had significant hearing loss.

**Bansal et al**[13] - Transient evoked otoacoustic emissions in hearing screening programs: protocol for developing countries – The objective of this program was to formulate a protocol for infant hearing screening in developing countries enabling it to be later incorporated into their national deafness screening programs. 2659 infants in the age range of 0-3 months were included in study. They were divided into 3 groups with age range between 0-1, 1-2and 2-3 months of age. All were subjected to transient evoked otoacoustic emission (TEOAE) for hearing screening. Those who failed first screening were followed up after 1-month.

Infants who had failed the second screening underwent Brainstem Evoked Response Audiometry (BERA). This study highlighted that delayed hearing screening at 3 months of age would considerably decrease the number of false positive cases avoiding unnecessary investigations and wastage of resources making the universal neonatal hearing screening within 48 h of life impractical for developing countries.Combining this delayed hearing screening with the 3rd dose of universal immunization program would constitute a viable, feasible and universal hearing screening program, which can be drafted into national deafness programs of the developing countries.

**Heinemann et al**[14] – Conducted a study to find the cost effectiveness of newborn hearing with different instruments and found that two step screening(first with OAE and then with BERA) was most cost effective.

**Finckh Kramer et al**[5]– Conducted a prospective study on the prevalence of hearing impairment in an at-risk neonatal intensive care unit (NICU) population.

**Sharma et al. and Dorman et al**[25]. have shown that neural plasticity within the auditory system begins to decline after approximately 3.5 years of

age and that earlier intervention tends to result in normal or near-normal central auditory function.

**Philips et al**.[25] determined that earlier screenings led to more successful outcome among children who were diagnosed with profound hearing loss and promptly fitted with cochlear implants.They concluded that this earlier intervention resulted in improved auditory receptivs skills.

**Apuzzo and Yoshinaga-Itano**[22] found that "infants who are identified with hearing loss early have a distinct advantage over their later identified peers" and "identification and intervention begun before 2-1/2 years benefits all infants with hearing loss, regardless of hearing impairment. This benefit is especially evident for subjects who were identified by two months of age."

**White and Maxon**[16] found universal newborn hearing screening to be more cost-effective than screening targeted only to infants considered high risk.

**1993** National Institute of Health (NIH) Consensus Development Panel on Early Identification of Hearing Impairment in Infants and Young Children suggested Universal screening is superior to a hearing protocol that screens only "high-risk" newborns because the high-risk protocol identifies

only 50 percent of hearing-impaired infants.The preferred model for infant screening should be two-stage beginning with an otoacoustic emissions test (OAE) and should be followed by an auditory brainstem response test (ABR) for all infants who fail the OAE test[18].

**Kurt et al** [8]– Stated that OAE testing can be accomplished easily in a normal newborn nursery with an acceptable false-positive rate when a two-stage approach is used.

**Kittrell et al**[17]revealed that average age of identification of hearing impairment was 20.2 months and average age of initial amplification was 31.7 months and two years after universal screening implementation,the everage age at diagnosis has improved to 3.8 months of age.

**Karen Jo Doyle et al** compared pass rates for two different hearing screening methods in well newborns as a function of age. Hearing screening tests were performed on 400 ears in 200 healthy newborn infants at the University of California-Irvine Medical Center. There was no significant difference in the AABR pass rate for infants aged 0–24 h of age as compared with infants aged >24 h.

However, the OAE pass rate improved significantly in infants >24 h compared with the group aged 0–24 h ($P < 0.01$).

**M D Mohd Khairi et al**[15] conducted 2 stage hearing assessment in 401 at risk neonates and concluded that mechanical ventilation of more than 5 days was not an independent risk factor for hearing impairment.

# AIM AND OBJECTIVES

## AIM :

To assess the hearing status of newborns admitted in our NICU with risk factors for hearing impairment.

## OBJECTIVES :

To set the new screening criteria for hearing assessment for babies with high risk factors, admitted in **NICU** of Government Rajaji Hospital,Madurai.

# MATERIALS AND METHODS

a)**Study Design :**

Prospective study

b**) Study Place :**

Neonatal intensive care unit (NICU), Institute of child health and research centre, Government Rajaji Hospital, Madurai- 20.

c) **Study Period :**

October 2009 to October 2010

d) **Study Population :**

Babies admitted in NICU of Government Rajaji Hospital, Madurai.

e) **Inclusion Criteria :**

All newborns with the following risk factors were enrolled for study.

1. Birth weight less than 1,500 grams (3.3 lbs.).
2. Apgar scores of 0- 4 at 1 minute ; 0-6 at 5 minutes.
3. Hyperbilirubinemia at a serum level requiring exchange transfusion
4. Ototoxic medications – Aminoglycosides and Loop diuretics used for >5 days
5. Mechanical ventilation lasting 5 days or longer.

6. Bacterial meningitis.
7. In utero infections by TORCH group of organisms.
8. Craniofacial anomalies, including those with morphological abnormalities of the pinna and ear canal.
9. Stigmata or other findings associated with a syndrome known to include a sensorineural and/or conductive hearing loss.
10. Family history of permanent childhood sensorineural hearing loss.

**METHODOLOGY**

All newborns with the above mentioned risk factors admitted in the NICU were screened for hearing impairment between October 2009 to October 2010. A total of 100 babies were subjected to OAE prior to discharge. Information collected regarding the selected cases were recorded in the master chart.

Babies with severe birth asphyxia (apgar 0-4 at 1 min and 0-6 at 5 min) were subjected to OAE after they met the discharge criteria Babies with hyperbilirubinemia were subjected to OAE after exchange transfusion and phototherapy reduced bilirubin to safe levels as per standard protocol.

Meningitis was diagnosed based on standard guidelines( CSF cell count and biochemical analysis) and babies were screened after they were completely treated.

Preterm babies with Very Low Birth Weight (Birth weight between 1000g to 1400g), babies who received ototoxic drugs and babies who were on ventilator were screened after they met the discharge criteria.

Two babies with craniofacial malformation were screened. One baby had defects in both eyes( microophthalmos, microcornea in left eye and blurred disc margin and optic nerve head dysplasia in the right side) and ear(microtia left side).CT scan of the baby showed contracted left eye with calcified focus in globe. Another baby had bilateral microtia with atresia of external auditory canal.

A baby was suspected as TORCH infection based on clinical findings (hepatosplenomegaly,petechiae,purpura and elevated direct bilirubin) and was screened on the day of discharge.

The initial examination was carried out by means of **OTO ACOUSTIC EMISSIONS (OAE)**.If the initial screening is normal. then the hearing is presumed to be normal and the neonate is advised follow up

every 6 months upto 3 years. If the initial screening is abnormal, then the newborn is subjected to **SECOND** screening again by **OAE** after one month**.** If the second screening is normal, then the hearing is presumed to be normal and the baby is advised follow up every 6 months upto 3 years.If the second screening is abnormal,then the baby is subjected to **AUTOMATED AUDITORY BRAINSTEM RESPONSE** (**AABR).**Based on the results of AABR, early intervention is done.

# OBSERVATION, ANALYSIS AND RESULTS

During this study,100 high risk babies were subjected to OAE testing. The age of the study group ranged between 3 days to 90 days.55 babies(55%) were male and 45 babies(45%) were female. The gestational age of the study group ranged between 30 to 38 weeks. Birth weight varied between 1000g and 3800g. 2 babies dropped out after the first screening test. At the end of two stage screening test,95 babies(96.9%) had normal hearing and 3 babies(3.1%) had hearing impairment(2 babies were male and 1 was female).Of the 3 babies, 2 babies had severe birth asphyxia and 1 baby had craniofacial malformation.

| | |
|---|---|
| Total babies screened initially by OAE | **:** 100 |
| Total babies who passed first screening by OAE | **:** 84(84%) |
| Total babies who failed first screening by OAE | **:** 16(16%) |
| Drop outs after first screening | **:** 02(12.5%) |
| Total babies subjected to second screening by OAE | **:** 14(87.5%) |
| Total babies who passed after second screening by OAE | **:** 12(85.7%) |
| Total babies who failed after second screening by OAE | **:** 02(14.3%) |

| | |
|---|---|
| Babies for whom OAE could not be done due to craniofacial malformation | : 01(1%) |
| Total number of babies for whom AABR is done | : 03(3.1%) |

3 babies were diagnosed to have hearing impairment out of 98 high risk babies ( mean = 0.03, S.D = 0.17). The incidence rate is 3.1% which is similar to other studies done (2.5 -10%).

## TABLE - 4

## RISK FACTORS SCREENED

| RISK FACTOR | MALE | FEMALE | PERCENTAGE |
|---|---|---|---|
| SEVERE BIRTH ASPHYXIA | 28 | 24 | 52 |
| VLBW | 10 | 10 | 20 |
| MENINGITIS | 06 | 04 | 10 |
| HYPERBILIRUBINEMIA | 04 | 04 | 08 |
| OTOTOXIC DRUGS | 03 | 01 | 04 |
| VENTILATED BABIES | 02 | 01 | 03 |
| CRANIOFACIAL ANOMALY | 02 | 00 | 02 |
| STIGMATA OF TORCH | 00 | 01 | 01 |

## TABLE – 5

## STUDY POPULATION

| | |
|---|---|
| ENROLLED | 100 |
| COMPLETED FOLLOW UP | 98 |
| DROP OUTS | 02 |

## TABLE-6

## SEX DISTRIBUTION OF SCREENEED INFANTS

| SEX | NUMBER | PERCENTAGE |
|---|---|---|
| MALE | 55 | 55% |
| FEMALE | 45 | 45% |
| TOTAL | 100 | 100% |

**TABLE - 7**

**SCREENING OF INDIVIDUAL RISK FACTORS**

**SEVERE BIRTH ASHYXIA**

| **FIRST SCREENING BY OAE** | | **SECOND SCREENING BY OAE** |
|---|---|---|
| 52 | TOTAL BABIES SCREENED | 06 |
| 45 | NORMAL HEARING | 04 |
| 02 | IMPAIRMENT IN ONE EAR | 01 |
| 05 | IMPAIRMENT IN BOTH EARS | 01 |

Of the 52 babies with birth asphyxia,45 babies passed the first screening. 2 babies had impairment in 1 ear and 5 babies had impairment in both ears. These 7 babies were planned second screening by OAE after one month. Of these 7 babies, 4 babies passed the second screening. 1 baby with impairment in both ears dropped out and 2 babies failed. Those 2 babies were subjected for AABR.

## TABLE - 8

## VERY LOW BIRTH WEIGHT

| FIRST SCREENING BY OAE | | SECOND SCREENING BY OAE |
|---|---|---|
| 20 | TOTAL BABIES SCREENED | 04 |
| 16 | NORMAL HEARING | 04 |
| 03 | IMPAIRMENT IN ONE EAR | 00 |
| 01 | IMPAIRMENT IN BOTH EARS | 00 |

Of the 20 babies with very low birth weight,16 babies passed the first screening.3 babies had impairment in 1 ear and 1 baby had impairment in both ears. All these 4 babies were subjected to second screening by OAE after one month. All the 4 babies who failed in the first screening passed when subjected to second screening by OAE after 1 month and had normal hearing.

**TABLE - 9**

**MENINGITIS**

| **FIRST SCREENING BY OAE** | | **SECOND SCREENING BY OAE** |
|---|---|---|
| 10 | TOTAL BABIES SCREENED | 03 |
| 07 | NORMAL HEARING | 03 |
| 02 | IMPAIRMENT IN ONE EAR | 00 |
| 01 | IMPAIRMENT IN BOTH EARS | 00 |

Of the 10 babies with meningitis,7 babies passed the first screeniong.2 babies had impairment in 1 ear and 1 baby had impairment in both ears. All these 3 babies were subjected to second screening by OAE after one month. All the 3 babies who failed in the first screening passed when subjected to second screening by OAE after 1 month.

**TABLE - 10**

**HYPERBILIRUBINEMIA REQUIRING EXCHANGE TRANSFUSION**

| **FIRST SCREENING BY OAE** | | **SECONDSCREENING BY OAE** |
|---|---|---|
| 08 | TOTAL BABIES SCREENED | 01 |
| 06 | NORMAL HEARING | 01 |
| 01 | IMPAIRMENT IN ONE EAR | 00 |
| 01 | IMPAIRMENT IN BOTH EARS | 00 |

Of the 8 babies, for whom exchange transfusion was done for hyperbilirubinemia, 6 babies passed the first screeniong.1 baby had impairment in 1 ear and 1 baby had impairment in both ears. Baby which failed in both ears dropped out. Other baby was subjected to second screening by OAE after one month and the baby passed the second screening.

Baby which failed in both ears dropped out and the other baby passed when subjected to second screening and had normal hearing.

## TABLE - 11

## OTOTOXIC DRUGS

## FIRST SCREENING BY OAE

| | |
|---|---|
| TOTAL BABIES SCREENED | 04 |
| NORMAL HEARING | 04 |
| HEARING IMPAIRMENT | 00 |

4 babies who received ototoxic drugs were screened and all the 4 babies passed the first screening by OAE.

## TABLE – 12

## VENTILATED BABIES

## FIRST SCREENING BY OAE

| | |
|---|---|
| TOTAL BABIES SCREENED | 03 |
| NORMAL HEARING | 03 |
| HEARING IMPAIRMENT | 00 |

Of the 3 babies who were ventilated for 5 days,all the 3 babies passed the first screening by OAE.

**TABLE – 13**

**CRANIOFACIAL MALFORMATION**

**FIRST SCREENING BY OAE**

| | |
|---|---|
| TOTAL BABIES SCREENED | 01 |
| NORMAL HEARING | 01 |
| HEARING IMPAIRMENT | 00 |

2 babies had craniofacial malformation.1 baby was directly subjected to AABR because OAE could not be done due to atresia of auditory canal. The other baby passed the first screening by OAE.

**TABLE – 14**

**TORCH INFECTION**

**FIRST SCREENING BY OAE**

| | |
|---|---|
| TOTAL BABIES SCREENED | 01 |
| NORMAL HEARING | 01 |
| HEARING IMPAIRMENT | 00 |

1 baby suspected as TORCH was screened and the baby passed the first screening by OAE**.**

## TABLE – 15

## AABR FOR FAILED SECOND SCREENING TEST

| RISK FACTOR | NO OF CASES AABR DONE | FINAL HEARING STATUS |
|---|---|---|
| SEVERE BIRTH ASPHYXIA | 02 | ABNORMAL |
| CRANIOFACIAL MALFORMATION | 01 | ABNORMAL |

3 babies were subjected to AABR and it was abnormal in all three babies.Hearing aids have been fitted with the help of ENT surgeon for 2 babies and 1 baby is awaiting hearing aid as it is only 3 months old.

## TABLE – 16

## FINAL OUTCOME OF SCREENED INFANTS

| OUTCOME | NUMBER OF CASES |
|---|---|
| NORMAL HEARING | 95 |
| HEARING IMPAIRMENT | 03 |

## TABLE - 17

## SEX DISTRIBUTION OF BABIES WITH HEARING IMPAIRMENT

| SEX | NUMBER |
|---|---|
| MALE | 2 |
| FEMALE | 1 |
| TOTAL | 3 |

## TABLE – 18

## FINAL OUTCOME OF BABIES WITH RISK FACTORS

| RISK FACTOR | TOTAL CASES | NORMAL HEARING | HEARING IMPAIRMENT |
|---|---|---|---|
| SEVERE BIRTH ASPHYXIA | 51 | 49 | 2 |
| VLBW | 20 | 20 | 0 |
| MENINGITIS | 10 | 10 | 0 |
| HYPERBILIRUBINEMIA | 07 | 07 | 0 |
| OTOTOXIC DRUGS | 04 | 04 | 0 |
| VENTILATED BABIES | 03 | 03 | 0 |
| CRANIOFACIAL ANOMALY | 02 | 01 | 1 |
| STIGMATA OF TORCH | 01 | 01 | 0 |

## TABLE 19

## CHI SQUARE TABLE

| RISK FACTOR | NORMAL HEARING | HEARING IMPAIRMENT | TOTAL CASES |
|---|---|---|---|
| SEVERE BIRTH ASPHYXIA | 49 | 2 | 51 |
| VLBW | 20 | 0 | 20 |
| MENINGITIS | 10 | 0 | 10 |
| HYPERBILIRUBINEMIA | 07 | 0 | 07 |
| OTOTOXIC DRUGS | 04 | 0 | 04 |
| VENTILATED BABIES | 03 | 0 | 03 |
| CRANIOFACIAL ANOMALY | 01 | 1 | 02 |
| STIGMATA OF TORCH | 01 | 0 | 01 |

Chi Square test has been applied to find the statistical significance of outcome of risk factors. Chi Square value is 16.40.The p value is 0.002.There is statistically significant difference among risk factors.

# DISCUSSION

Ideally all newborns should be screened for hearing loss prior to discharge from hospital where they were born. However in developing country like ours with limited resources,this is not always possible. Hence newborns with risk factors for hearing loss should **atleast** be screened.

The incidence of hearing impairment in high risk neonates according to different statistics is 2.5% - 10%.[10] In this study, the incidence is 3.1% which is similar to other studies.

100 babies were subjected to OAE testing.**16( 16%)** babies failed after the first screening.They include **7** babies with birth asphyxia,**4** babies with very low birth weight,**3** babies who were treated for meningitis and **2** babies for whom exchange transfusion was done for hyperbilirubinemia.2(12.5%)babies dropped out after the first screening test.12 out of 14(85.7%) babies passed the second screening by OAE.2 out of 14 babies(14.3%) failed after second screening by OAE and were subjected to AABR. Totally 3 babies were subjected to AABR( 2 babies who failed screening along with another baby who was directly subjected to AABR as the baby had atresia of external auditory canal) and all the 3 babies had abnormal AABR(3.06%). All 3 babies had profound hearing loss.

## BIRTH ASPHYXIA

52 babies with severe birth asphyxia were screened by OAE. 7 (13.5%) babies failed the first screening by OAE. Of these, 5 babies had impairment in both ears and 2 babies had impairment in one(left) ear. Both of them had associated Congenital heart disease.4 babies passed when subjected to second screening; 1 baby dropped out and finally **2** (3.9%) babies with birth asphyxia had hearing impairment at the end of 2nd screening.

A study by **Nagapoornima et al**[4] who screened 51 babies with severe birth asphyxia and identified hearing impairment in **1** baby. **Christine Ohl et al**[7] screened 12 babies with severe birth asphyxia and identified 4 babies with hearing impairment which is much higher than our study.

## VERY LOW BIRTH WEIGHT

Twenty babies with very low birth weight were by screened by OAE. 4 (20%) babies failed the first screening by OAE. Of these 4, 3 babies had impairment in 1 ear and 1 baby had impairment in both the ears.All the 4 babies passed when subjected to second screening by OAE. A study by **Christine Ohl et al**[7] who showed very low birth weight is not a risk factor for hearing impairment as in our study in which babies with VLBW had normal hearing. A study by **Finckh Kramer U et al**[5] and **Hess M et al**[6] also

concluded that VLBW was not a predictor of hearing impairment as in our study.

## MENINGITIS

Ten babies who were treated for meningitis were by screened by OAE.3 (30%) babies failed the first screening by OAE. Of these 3, 2 babies had impairment in 1 ear and 1 baby had impairment in both the ears. All the 3 babies passed when subjected to second screening by OAE.

**Nagapoornima et al**[4] screened 14 babies with meningitis but none had hearing impairment as in our study.

## EXCHANGE TRANSFUSION

Eight babies who underwent exchange transfusion for hyperbilirubinemia were by screened by OAE.2 (25%) babies failed the first screening by OAE.1 baby dropped out after the first screening.The other baby had impairment in left ear and passed when subjected to second screening by OAE.**Nagapoornima et al**[4] screened 38 babies with severe hyperbilirubinemia requiring exchange transfusion but none had hearing impairment as in our study. This is due to early identification and effective management of hyperbilirubinemia.

## OTOTOXIC DRUGS

Four babies who received ototoxic drugs for septicemia were screened by OAE and all the 4 babies passed the screening test. **Finckh Kramer U et al**[5] concluded that aminoglycosides are not an important risk factor.Similar results were obtained by **Hess M et al**[6] and our study also showed aminoglycosides are not a risk factor for hearing impairment

## VENTILATED BABIES

Three babies who were ventilated for birth asphyxia and sepsis were screened and all the 3 passed the first screening by OAE.

**M D Mohd Khairi et al**[15] conducted 2 stage hearing assessment in 401 at risk neonates and concluded that mechanical ventilation of more than 5 days was not an independent risk factor for hearing impairment.

## CRANIOFACIAL MALFORMATION

Two babies with craniofacial malformation were included in our study.1 baby passed the first screening by OAE.Other baby was directly subjected to AABR as the baby had bilateral atresia of external auditory canal and the baby had abnormal AABR**.**

**Nagapoornima et al**[4] screened 24 babies with craniofacial malformation but none had hearing impairment in contrast to our study in

which 1 of the 2 (50%) babies with craniofacial malformation had hearing impairment.

## TORCH INFECTION

One baby was suspected as TORCH infection clinically and was screened and the baby passed the first screening by OAE.

**Nagapoornima et al**[4] screened 6 babies with TORCH infection but none had hearing impairment as in our study.

Out of 16 babies who failed after the first screening,2 dropped out. Of the remaining 14 babies, 12 babies passed when subjected to second screening. Finally 2 babies failed after 2nd screening. These 2 babies along with the baby who had craniofacial malformation were subjected to AABR and all the 3 babies had abnormal AABR.

So in our study of **high risk** screening, 3 babies had hearing impairment (3.1%) out of 98and it is higher than the incidence of study by **Nagapoornima et al**[4] who identified 3 out of 279 high risk babies(1.07%).

**Christine Ohl et al**[4] screened 1461at risk babies among whom 4.55% were diagnosed as deaf which is higher than our study.

**Finckh Kramer U et al**[5] screened 1062 at risk neonates and identified hearing Impairment in 1.3% which is lower than our study.

**Sayed Hossein Fakhraee et al**[24] screened 150 high risk infants of whom 42(28%) had different levels of hearing impairment.

# CONCLUSION

- In this study of 100 high risk babies, 3 babies(3.1%) had hearing impairment(profound hearing loss).
- Of the 8 risk factors screened, only 2 risk factors (severe birth asphyxia and craniofacial malformation) were associated with hearing impairment.
- 3.9%(2 out of 51) of babies with severe birth asphyxia and 50%(1 out of 2) of babies with craniofacial malformation had hearing impairment.
- Hearing impairment is not seen in VLBW infants, Meningitis, Hyperbilirubinemia, Ventilated babies and those who received Ototoxic drugs probably due to early and effective management.
- Hence early identification and intervention will allow deaf and hard of hearing children to develop language skills during a period of neural plasticity that would otherwise be forfeited, banishing them into a world of social isolation and educational malaise.

# LIMITATIONS

- Our study focused on high risk infants who constitute only 50% of all neonates with hearing loss. The other 50% will remain undetected at birth by this approach.
- All high risk babies require hearing assessment every 6 months upto 3 years which could not be done in our study.

# RECOMMENDATIONS

Developing countries like India must take initiatives to implement newborn hearing screening programme. Initially a centralized screening facility can be established to implement this program.

- Each District Hospital should run a program and the Audiologist should function as program co-ordinator.
- All children born in the district should be screened at birth, or, within a month's time. Primary health centers and community health centers should make arrangements for referral. Cost effective behavioural observation methods using calibrated noise making toys may be taught to anganwadi workers and may be advised to refer to higher centres if needed.
- New-borns who fail on screening, should be given a diagnostic test and proper interventions within 3 months.
- Those who have high-risk for hearing loss should be followed up at intervals of 6 months even if they are cleared at the screening.

If resources are limited, then one could focus initially on high risk neonates and gradually implement universal screening.

The message is **– "Don't take a chance, have a hearing testing done in all newborns".**

# Brainstem Auditory Evoked Response

**Medical Record Number :** 16737 **Date:** 12.10.10

**Name :** J. Sivasakthi

**Age / Sex :** 3 months ; Female

**Results:**

Both Ears : Peak 'V' couldn't be observed even at 99 dBnHL

**Impression:**

Bilateral Severe to Profound Hearing Loss.

**Recommendation:**

- Cochlear Implant Counselling
- Hearing aid Trial and fitting
- Speech and Language therapy
- Follow up.

**Baiju. K.S**
**Audiologist**

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**Baiju. K.S**
**Audiologist**

| S.No | NAME | AGE (IN DAYS) | SEX | BIRTH WT (IN GRAMS) | APGAR AT 1 & 5 MIN | EXCHANGE TRANSFUSION | SEVERE BIRTH ASPHYXIA | MENINGITIS | VENTILATED | CRANIOFACIAL ANOAMLY | OTOTOXIC DRUGS | TORCH | OAE RESULTS RIGHT | OAE RESULTS LEFT | AABR RESULTS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | B/O Perinbanayagi | 6 | F | 1000 | 4/10;6/10 | N | N | N | N | N | N | N | P | P | |
| 2 | B/O Jeyakodi | 13 | F | 1240 | 5/10;7/10 | N | N | N | N | N | N | N | P | P | |
| 3 | B/O Kesavapriya I | 11 | M | 1400 | 5/10;7/10 | N | N | N | N | N | N | N | P | P | |
| 4 | B/O Kesavapriya II | 11 | M | 1100 | 5/10;7/10 | N | N | N | N | N | N | N | P | P | |
| 5 | B/O Dhanabackiyam | 7 | F | 1400 | 4/10;6/10 | N | N | N | N | N | N | N | P | P | |
| 6 | B/O Muthukili | 14 | M | 1100 | 5/10;7/10 | N | N | N | N | N | N | N | P | P | |
| 7 | B/O Sathipriya | 12 | M | 1200 | 5/10;7/10 | N | N | N | N | N | N | N | P | P | |
| 8 | B/O Sathya | 16 | M | 1300 | 5/10;7/10 | N | N | N | N | N | N | N | P | P | |
| 9 | B/O Alagu | 5 | F | 1100 | 4/10;7/10 | N | N | N | N | N | N | N | P | P | |
| 10 | B/O Jeyalakshmi | 5 | F | 1200 | 5/10;7/10 | N | N | N | N | N | N | N | P | P | |
| 11 | B/O Seeni Syedammal | 7 | F | 1100 | 4/10;6/10 | N | N | N | N | N | N | N | P | P | |
| 12 | B/O Sureka | 21 | F | 1000 | 5/10;7/10 | N | N | N | N | N | N | N | P | P | |
| 13 | B/O Alagammai | 17 | M | 1250 | 5/10;8/10 | N | N | N | N | N | N | N | P | P | |
| 14 | B/O Roopa | 8 | F | 1200 | 4/10;6/10 | N | N | N | N | N | N | N | P | P | |
| 15 | B/O Raghavi | 6 | F | 1100 | 5/10;7/10 | N | N | N | N | N | N | N | P | P | |
| 16 | B/O Anusha Devi | 4 | M | 1400 | 5/10;7/10 | N | N | N | N | N | N | N | P | P | |
| 17 | B/O Parimalam | 8 | F | 1400 | 4/10;6/10 | N | N | N | N | N | N | N | P | P | |
| 18 | B/O Muniyammal | 12 | M | 1400 | 3/10;7/10 | N | N | N | N | N | N | N | P | P | |
| 19 | B/O Poonkodi | 12 | M | 1250 | 4/10;7/10 | N | N | N | N | N | N | N | P | P | |
| 20 | B/O Manjula | 17 | M | 1200 | 4/10;7/10 | N | N | N | N | N | N | N | P | P | |
| 21 | B/O Ramila | 7 | M | 2300 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 22 | B/O Maheshwari | 6 | F | 2700 | 2/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 23 | B/O Gopala Krishnammal | 6 | M | 3000 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 24 | B/O Karthigai Selvi | 4 | F | 3100 | 2/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 25 | Jothi Eswar | 5 | M | 1900 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 26 | B/O Rathinam | 8 | M | 2700 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 27 | B/O Mareeswari | 5 | F | 2400 | 2/10;5/10 | N | Y | N | N | N | N | N | R | R | |
| 28 | Krithik Roshan | 4 | M | 2600 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 29 | B/O Muthumari | 6 | M | 2300 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 30 | B/O Mariyam | 5 | M | 2200 | 2/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 31 | Ali Akbar | 11 | M | 1800 | 2/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 32 | B/O Mehar Nisha | 7 | F | 3000 | 2/10;4/10 | N | y | N | N | N | N | N | P | P | |
| 33 | B/O Jegar Banu | 6 | F | 2700 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 34 | Sivasakthi | 8 | F | 2600 | 2/10;5/10 | N | Y | N | N | N | N | N | R | R | A |
| 35 | B/O Sivashankaraselvi | 6 | F | 2400 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 36 | B/O Kavitha | 7 | M | 2800 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 37 | B/O Revathy | 8 | M | 2900 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 38 | B/O Nagarathinam | 6 | F | 3600 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 39 | B/O Rajalakshmi | 7 | F | 3100 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 40 | B/O Pavithra | 8 | M | 2400 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 41 | B/O Sasikala | 8 | M | 2300 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 42 | B/O Bhavani | 4 | F | 2700 | 2/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 43 | B/O Karthiga | 6 | M | 2600 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 44 | B/O Sharmila | 5 | F | 2400 | 3/10;5/10 | N | y | N | N | N | N | N | P | P | |
| 45 | B/O Asha | 5 | M | 2400 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 46 | B/O Shanmugapriya | 6 | M | 3000 | 2/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 47 | B/O Podhumponnu | 7 | F | 3100 | 2/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 48 | B/O Jansirani | 6 | F | 3300 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 49 | B/O Sivapriya | 5 | F | 3500 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 50 | Mayathevan | 7 | M | 2100 | 3/10;5/10 | N | Y | N | N | N | N | N | R | R | A |
| 51 | B/O Basheera Banu | 7 | F | 2100 | 2/10;4/10 | N | y | N | N | N | N | N | P | P | |
| 52 | B/O Umamaheshwari | 6 | F | 2600 | 2/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 53 | B/O Rajalakshmi | 6 | M | 2600 | 2/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 54 | B/O Kalaivani | 6 | M | 2400 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 55 | B/O Kokila | 5 | M | 2800 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 56 | B/O Poomari | 5 | F | 2900 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 57 | B/O Pandimeenakshi | 5 | M | 2900 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 58 | B/O Saritha | 6 | F | 3000 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 59 | B/O Saraswathi | 6 | F | 2100 | 2/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 60 | B/O Anandhi | 5 | M | 2000 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 61 | B/O Dhanabackiyalakshmi | 5 | F | 2300 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 62 | Yogesh kumar | 5 | M | 2100 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 63 | B/O Kattuarani | 5 | M | 2000 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 64 | B/O Punitha | 7 | M | 2500 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 65 | B/O Umamaheshwari | 7 | F | 2700 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 66 | B/O Shobana | 6 | F | 2800 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 67 | B/O Pandeeswari | 8 | F | 2500 | 2/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 68 | B/O Sathyapriya | 7 | F | 2800 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 69 | B/O Kumareswari | 7 | M | 2600 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 70 | B/O Revathy | 6 | M | 2800 | 3/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 71 | B/O Amsavalli | 9 | M | 2100 | 2/10;4/10 | N | Y | N | N | N | N | N | P | P | |
| 72 | B/O Anis Fathima | 6 | M | 2000 | 2/10;5/10 | N | Y | N | N | N | N | N | P | P | |
| 73 | B/O Sathish rani | 17 | M | 3800 | 6/10;8/10 | N | N | Y | N | N | N | N | P | P | |
| 74 | B/O Bargath Nisha | 7 | F | 3600 | 6/10;8/10 | N | N | Y | N | N | N | N | P | P | |
| 75 | B/O Banumathi | 14 | F | 3400 | 7/10;8/10 | N | N | Y | N | N | N | N | P | P | |
| 76 | B/O Renganayaki | 11 | M | 3100 | 3/10;6/10 | N | Y | Y | N | N | N | N | P | P | |
| 77 | B/O Manjula Devi | 23 | M | 3400 | 3/10;6/10 | N | Y | Y | N | N | N | N | P | P | |
| 78 | B/O Shanmugavalli | 3 | M | 2750 | 7/10;8/10 | N | N | Y | N | N | N | N | P | P | |
| 79 | B/O Sangeetha | 5 | M | 2600 | 7/10;8/10 | N | N | Y | N | N | N | N | P | P | |
| 80 | B/O Nagalakshmi | 11 | M | 3000 | 7/10;8/10 | N | N | Y | N | N | N | N | P | P | |
| 81 | B/O Hemalatha | 4 | F | 2800 | 7/10;8/10 | N | N | Y | N | N | N | N | P | P | |
| 82 | B/O Sulochana | 3 | M | 2400 | 7/10;8/10 | N | N | Y | N | N | N | N | P | P | |
| 83 | Madesh | 3 | M | 3000 | 3/10;5/10 | Y | Y | N | N | N | N | N | P | P | |
| 84 | B/O Devika | 3 | F | 2750 | 6/10;8/10 | Y | N | N | N | N | N | N | P | P | |
| 85 | B/O Revathy | 6 | M | 3100 | 6/10;8/10 | Y | N | N | N | N | N | N | P | P | |

| | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 86 | B/O Vallupriya | 4 | F | 2600 | 3/10;5/10 | Y | Y | N | N | N | N | N | P | P | |
| 87 | B/O Podhumani | 4 | F | 3000 | 6/10;8/10 | Y | N | N | N | N | N | N | P | P | |
| 88 | B/O Parameshwari | 4 | M | 2600 | 7/10;9/10 | Y | N | N | N | N | N | N | R | R | |
| 89 | B/O Nagajothi | 4 | M | 2800 | 7/10;9/10 | Y | N | N | N | N | N | N | P | P | |
| 90 | B/O Katheeja Banu | 3 | F | 3200 | 6/10;8/10 | Y | N | N | N | N | N | N | P | P | |
| 91 | B/O Sonia | 15 | M | 3200 | 6/10;8/10 | N | N | N | N | N | Y | N | P | P | |
| 92 | B/O Murugayee | 17 | M | 3100 | 6/10;8/10 | N | N | N | N | N | Y | N | P | P | |
| 93 | B/O Nagalakshmi | 13 | F | 3000 | 3/10;6/10 | N | Y | N | N | N | Y | N | P | P | |
| 94 | B/O Amsavalli | 10 | F | 2900 | 3/10;6/10 | N | Y | N | N | N | Y | N | P | P | |
| 95 | B/O Shanmugavalli | 26 | M | 2300 | 5/10;7/10 | N | N | N | Y | N | N | N | P | P | |
| 96 | Velmurugan | 24 | M | 2900 | 3/10;6/10 | N | Y | N | Y | N | N | N | P | P | |
| 97 | B/O Santhanalakshmi | 9 | F | 2500 | 3/10;5/10 | N | Y | N | Y | N | N | N | P | P | |
| 98 | Balamurugan | 21 | M | 3100 | 6/10;8/10 | N | N | N | N | Y | N | N | P | P | |
| 99 | Madhan Kumar | 25 | M | 2700 | 6/10;8/10 | N | N | N | N | Y | N | N | R | R | A |
| 100 | B/O Nagajothi | 15 | F | 2700 | 6/10;8/10 | N | N | N | N | N | N | Y | P | P | |

# Brainstem Auditory Evoked Response

**Medical Record Number :** 10519 **Date:** 27.01.2010

**Name :** M. Mayadevan

**Age / Sex :** 2 ½ months ; male

**Results:**

Both Ears: Peak V could not be observed till 99dBnHL

**Impression:**

Both Ears: Severe to Profound Hearing Loss

**Recommendation:**

- Hearing Aid Trial
- Follow up.

**Asha Manoharan**
**Audiologist**

# Audiological Report

**M.R No: 10519** **Date:** 27.01.2010

**Name :** M. Mayadevan

**Age / Sex:** 2 ½ months ; male

**Immittance Audiometry:**

Both Ears- **'A'** Type with reflexes absent
S/o Normal middle ear functioning.

**OAE:**

Both Ears **–** Emissions absent
S/o OHC dysfunctioning.

**ABR:**
Both Ears – Peaks could not be identified till 99dBnHL

**ASSR:**
Both Ears – Profound Hearing Loss

**Provisional Diagnosis:**

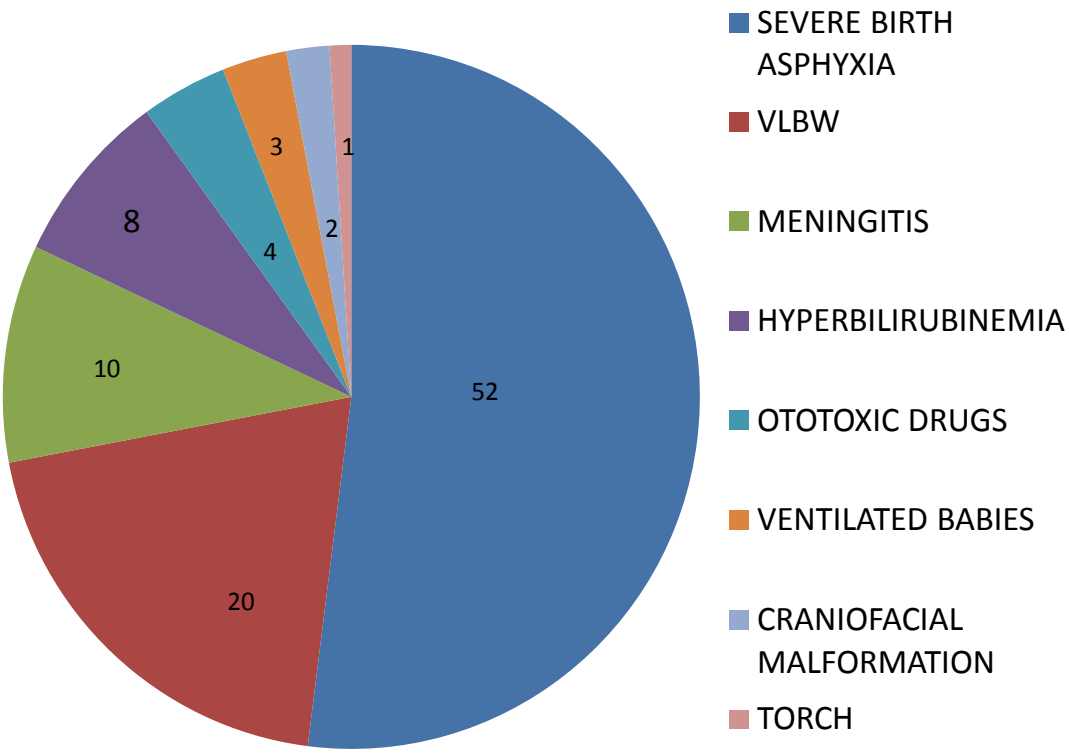
Bilateral profound hearing loss

**Recommendation:**

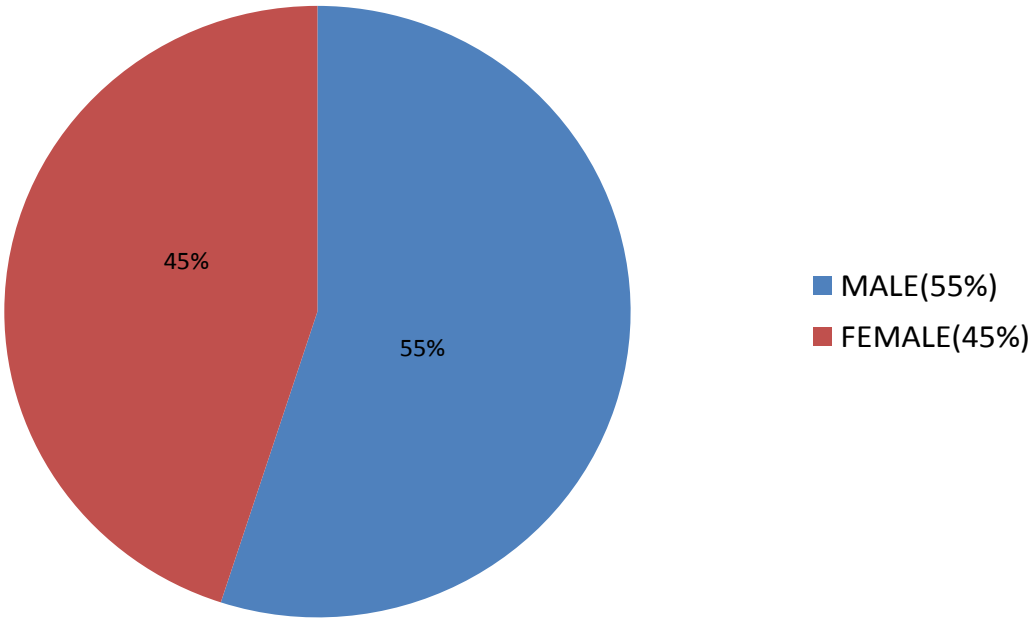
- Hearing Aid Trial and fitting
- Speech and language evaluation and therapy
- Special education
- Follow Up

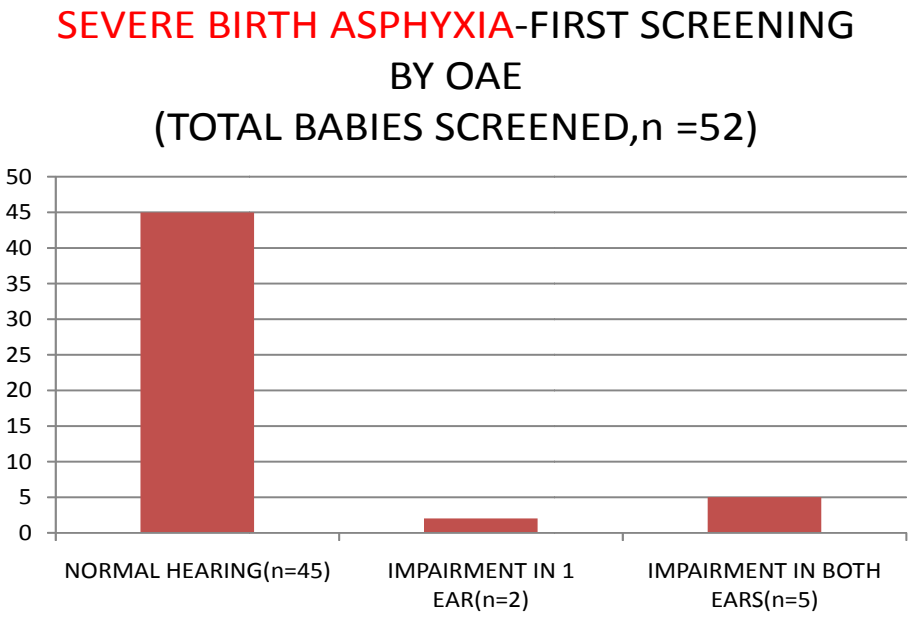
**Asha Manoharan**
**Audiologist**

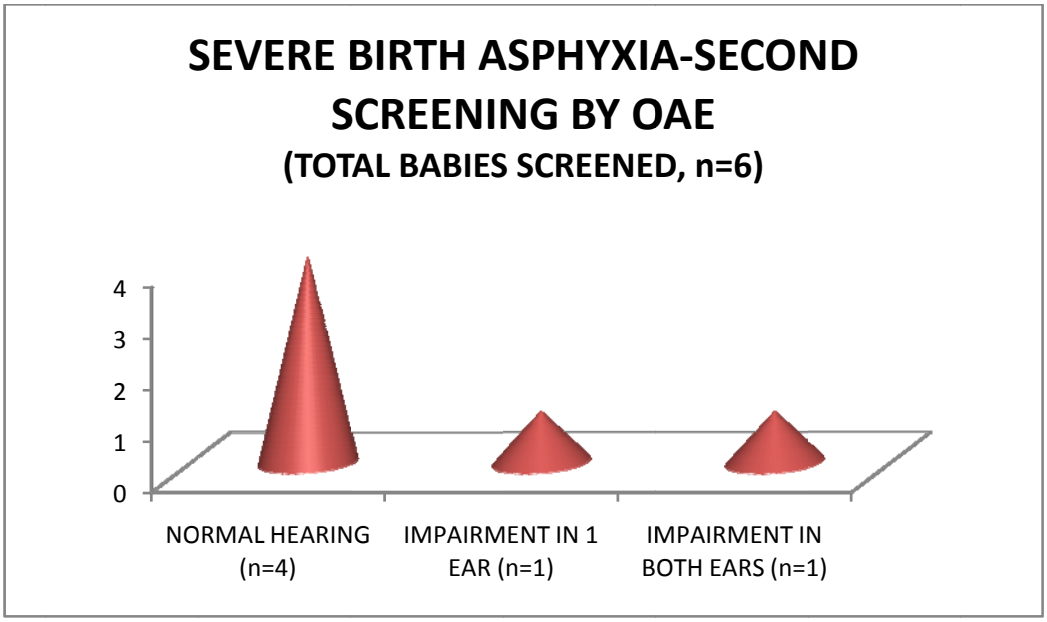
# RISK FACTORS SCREENED



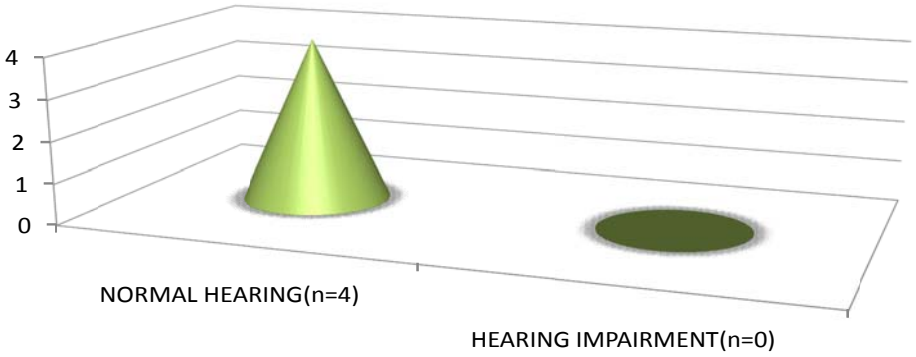
# SEX DISTRIBUTION OF SCREENED INFANTS



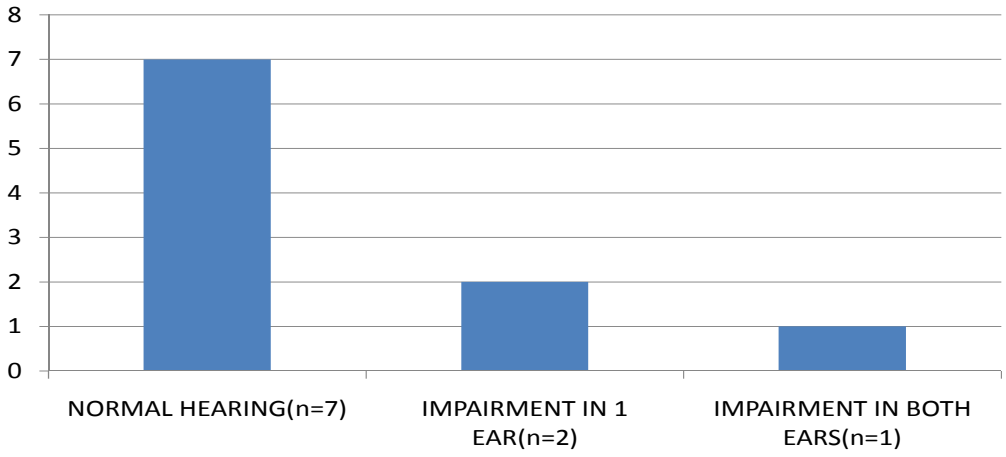

SEVERE BIRTH ASPHYXIA-FIRST SCREENING BY OAE
(TOTAL BABIES SCREENED,n =52)
50
45
40
35
30
25
20
15
10
5
0
NORMAL HEARING(n=45)
IMPAIRMENT IN 1 EAR(n=2)
IMPAIRMENT IN BOTH EARS(n=5)


SEVERE BIRTH ASPHYXIA-SECOND SCREENING BY OAE
(TOTAL BABIES SCREENED, n=6)
4
3
2
1
0
NORMAL HEARING (n=4)
IMPAIRMENT IN 1 EAR (n=1)
IMPAIRMENT IN BOTH EARS (n=1)

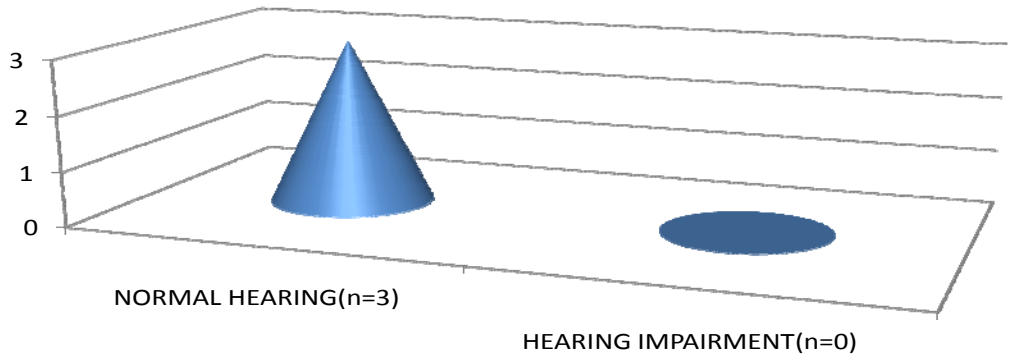
# VLBW-SECOND SCREENING BY OAE (TOTAL BABIES SCREENED,n=4)



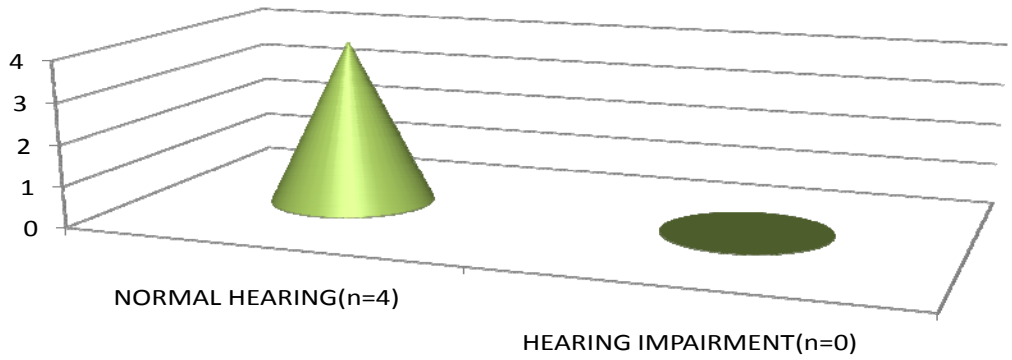
## MENINGITIS-FIRST SCREENING BY OAE (TOTAL BABIES SCREENED,n =10)



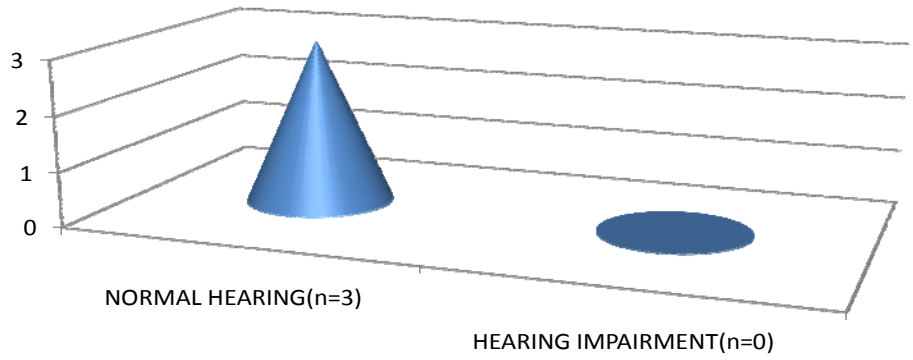

## MENINGITIS-SECOND SCREENING BY OAE (TOTAL BABIES SCREENED,n=3)



## OTOTOXIC DRUGS-FIRST SCREENING BY OAE (TOTAL BABIES SCREENED,n=4)




## VENTILATED BABIES-FIRST SCREENING BY OAE (TOTAL BABIES SCREENED,n=3)



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# PROFORMA

Name : I.P.NO. :

Age : Address :

Sex :

Date of birth :

Term / Preterm :

Birth Place :

Birth Weight :

Apgar at 1 & 5 min :

## RISK FACTORS

| | |
|---|---|
| Birth asphyxia | Y/N |
| VLBW | Y/N |
| Hyperbilirubinemia requiring exchange transfusion | Y/N |
| Meningitis | Y/N |
| Ototoxic drugs | Y/N |
| Mechanical Ventilation | Y/N |
| Craniofacial Malformation | Y/N |
| TORCH Infection | Y/N |
| Family h/o childhood hearing loss | Y/N |

## SCREENING METHOD BY OAE

| | RIGHT EAR | | LEFT EAR | |
|---|---|---|---|---|
| FIRSTSCREENING | PASS | REFER | PASS | REFER |
| SECONDSCREENING | PASS | REFER | PASS | REFER |

## CONFIRMATION BY AABR

| | RIGHT EAR | LEFT EAR |
|---|---|---|
| AABR | Normal/Abnormal | Normal/Abnormal |

RESULTS

# ABBREVIATIONS

| | |
|---|---|
| DCN | Dorsal Cochlear Nucleus |
| VCN | Ventral Cochlear Nucleus |
| NIH | National Institute of Health |
| NICU | Neonatal Intensive Care Unit |
| JCIH | Joint Committee on Infant Hearing |
| EHDI | Early Hearing Detection and Intervention |
| CDC | Centre for Disease Control |
| OAE | Oto Acoustic Emissions |
| AABR | Automated Auditory Brainstem Response |
| VLBW | Very Low Birth Weight |